EXHIBIT 9

EXPERT REPORT OF DR. GAIL D. STOCKMAN

A. Qualifications

1. I am Gail D. Stockman, MD, Ph.D. My address is 157 Creston Road, Kalispell, MT 59901.
2. I am licensed to practice medicine in Texas and Montana.
3. I am a Phi Beta Kappa graduate of the University of Texas in Austin. I received a Bachelor of Arts degree with high honors in microbiology and chemistry.
4. I have a Master of Arts degree from the University of California, Berkeley, in bacteriology and molecular biology.
5. I have a Ph.D. from the Experimental Biology department of Baylor College of Medicine in Houston, Texas.
6. From 1971 to 1974, I served on the faculties of Baylor College of Medicine and University of Texas M.D. Anderson Hospital and Tumor Research Institute, where I taught experimental design and supervised research projects.
7. During my research years, I authored or co-authored sixteen peer-reviewed journal articles and presented my research at multiple national and international conferences.
8. In 1976, I graduated medical school at Baylor College of Medicine with honors. I was elected to Alpha Omega Alpha, the national medical honor society.
9. I completed an internship and residency in Internal Medicine at the Baylor Affiliated Hospitals in Houston. I served six months as Chief Medical Resident at the Houston Veterans Administration Hospital. I then completed the clinical year of a

pulmonary fellowship at Baylor College of Medicine. I am a board-certified internist.

10. I practiced pulmonary and critical care medicine in Longview Texas from 1981 until April 2007 when I moved to Kalispell, MT to join the Rocky Mountain Heart and Lung Clinic as a pulmonologist.
11. My curriculum vitae are attached as Exhibit 1.
12. In the early 1990's, I completed a review of the world literature on asbestos-related diseases, beginning in 1898. I have subsequently testified at trial on a number of occasions regarding what could or should have been known at various time periods regarding asbestos-related disease.
13. During my practice of medicine in Longview, Texas, I diagnosed and cared for patients with asbestosis, lung cancer, and malignant mesothelioma in addition to chest injuries, chronic obstructive pulmonary disease, asthma, etc. In addition, I saw large volumes of patients for Independent Medical Evaluations between about 1995 and 2005. Most of these patients had been exposed to asbestos or silica or both.
14. Since 2008, I have provided pulmonary "second opinion reviews" based on medical records and chest x-rays and CT scans to the Libby Medical Program on approximately 25-30 patients of the CARD clinic. In addition, I have seen and examined twelve to fifteen patients for the Libby Medical Program, and I have evaluated and followed fifteen to twenty patients from Libby who were either self-referred or referred by their primary physicians.
15. I have been retained by the Grace asbestos claimants committee as a rebuttal witness to Dr. Alan Whitehouse. My fee is $400.00 per hour.

16. During the past four years, I have testified at one trial and have given eight depositions as follows:

10-25-05: *Sergio H. Enriquez, on behalf of the Estate of Maria G. Rodriguez, et al. V. Del Sol Medical Center,* et al; In the 327th Judicial District Court of El Paso County, Texas. (Deposition in a medical malpractice case.)

2-26-06: Cause No. 02-1132; *Denny Abernathy et al vs. ABB Lummus, Inc., et al;* In the 71st Judicial District Court of Harrison County, Texas. (Trial testimony with regard to a review of medical records.)

9-18-08: *Raymond Johnson V. Liberty Northwest Insurance Corporation*; WCC No. 2004-1092; Claim No. WC687-144471-00. (Deposition with regard to an independent medical evaluation.)

10-10-08: *Chester Goodwin v. A.W. Chesterton;* Cause No. RG08378296; Superior Court of Alameda County, California. (Deposition with regard to medical records review.)

10-24-08: *Robert and Geraldine Hilt v. Asbestos Defendants;* Docket No. 274561; San Francisco County Superior Court. (Deposition with regard to medical records review).

11-23-08: *Margaret Simpson v. Asbestos Defendants, et al.* Case No: CGC-070274941; San Francisco Superior Court. (Deposition with regard to medical records review).

12-12-08: *James Prough & Margaret Prough v. Allis Chalmers, et al;* Case No. BC389423; Los Angeles Superior Court. (Deposition with regard to medical records review).

12-12-08: *Jean Harlow-Biles v. Asbestos Defendants;* Case No. CGC-07-27403; San Francisco Superior Court. (Deposition with regard to medical records review.)

3-15-09: *Linda Strickland, et al. v. Advocate Mines, et al;* Case No. BC379088; Los Angeles Superior Court. (Deposition regarding medical records review.)

B. Asbestos Minerals

17. The term "asbestos" refers to a family of naturally occurring minerals that share properties of heat, chemical resistance, flexibility, and high tensile strength. Because of those desirable properties, they have been incorporated into as many as 3000 different products in our industrialized society.

16. There are two major mineralogic groups of asbestos fibers: serpentine and amphibole. Chrysotile (white asbestos) is the only serpentine fiber. Under the microscope, chrysotile fibers appear as flexible, spiral-shaped fibers. Chrysotile has been mined in Canada and used extensively in this country. There are two commercially important amphiboles: Crocidolite (blue asbestos) was mined in South Africa and Western Australia, and used primarily in Great Britain (although small amounts were used in this country). Under the microscope, crocidolite fibers are long, inflexible slender rods that more easily penetrate lung tissues, making them much more hazardous than the other types of asbestos. Because of its propensity for causing malignant mesothelioma, crocidolite is no longer mined or used commercially. Amosite (brown asbestos) is an amphibole that was used in production of insulation materials in this country, making up between 5-10% of some commercially used products.

Under the microscope, it is an inflexible rod that is thicker than crocidolite.

17. Tremolite is an amphibole that is not used commercially, but it is a frequent contaminant of other mineral ores, especially chrysotile asbestos, vermiculite (as in Libby), and talc.

C. Non-malignant Asbestos-Related Diseases

18. Studies from North America and Western Europe have indicated that virtually everyone in the general population has asbestos bodies (coated amphibole asbestos fibers) in their lungs. One study of the general population of Vancouver estimated that the lungs of the urban dwellers contained an average of 40,000 asbestos bodies with no evidence of asbestos-related disease (Churg A., textbook). The development of physical evidence of asbestos exposure or an asbestos-related disease depends in part on individual variation among humans (most likely genetic differences), since among workers with the same exposure, only a portion will develop asbestos-related disease.

19. Cigarette smoking has been repeatedly shown to increase the incidence of asbestos-induced pulmonary interstitial fibrosis (Lilis, 1986; Pearle, 1982; Delclos, 1990; and McMillan, 1980). Lillis, et al demonstrated that cigarette smoking led to a much earlier development of parenchymal opacities than was seen in nonsmokers. Human studies have demonstrated that cigarette smoking dramatically enhances retention of fibers of all types in the airway mucosa (Churg, 1995). Smoking was not found to result in a

statistically significant increase in diffuse pleural fibrosis. There is disagreement in the medical literature as to whether cigarette smoking increases the incidence of pleural plaques. McMillan (1980) found an increased prevalence of pleural plaques in British dockyard workers who were smokers or ex-smokers. Delclos (1990) did not find an increased prevalence of pleural plaques in a cohort of American end-product users (primarily insulators).

D. Asbestosis

20. ***Asbestosis*** is defined in all major textbooks and the vast majority of the medical literature as "bilateral diffuse interstitial fibrosis of the lungs caused by the inhalation of asbestos fibers." To quote Dr. Churg in his textbook, Pathology of Occupational Lung Disease, "this is the **only** condition to which the label ***asbestosis*** should be applied. Some authors include asbestos-related pleural diseases under the heading of asbestosis; ***this usage is to be avoided, since it lumps together a disparate set of diseases with different epidemiologic, pathogenetic, pathologic, and prognostic features.***" He goes on to say "use of the term *pleural asbestosis"* to refer to pleural lesions…is an unfortunate choice of words, since asbestos-associated benign pleural changes are mostly radiographic and pathologic findings that only infrequently have significant symptoms or functional consequences, whereas true asbestosis (i.e., interstitial fibrosis of the parenchyma) is frequently associated with functional impairment. For this reason *pleural lesions should never be called asbestosis."* After this statement, Dr. Churg cites the American Thoracic

Society consensus statement on the diagnosis of nonmalignant diseases related to asbestos, 1986.

21. The inappropriate use of the term "*asbestosis*" by Dr. Whitehouse and the CARD clinic to include benign pleural plaques and pleural thickening, and the comparison of the incidence of "*asbestosis*" in Libby with reports of asbestosis, accurately defined in other studies, has resulted in a gross inflation of the estimation of significant asbestos-related disease in the Libby population.

22. In a 2007 publication by Sullivan, a cohort of 1,830 workers who had mined, milled, and processed Libby vermiculite for a minimum of twenty years were studied though 2001. Nearly 47% of those workers had died by that time. The cause of death was determined from *death certificates* (which are notoriously inaccurate). Dr. Sullivan found that 5.3% of that population had reportedly died of "asbestosis," as reported by their attending physicians at the time of death. This death rate from "*asbestosis*" was higher than the 4% of deaths ascribed to asbestosis in a cohort of over 17,000 insulators with heavy exposures to asbestos, including amphibole asbestos (Selikoff, 1991). The very high incidence of asbestosis in the Libby population was seemingly confusing to Dr. Sullivan, and she spent about a page of her paper theorizing as to why the death rate from asbestosis in Libby was so high. In fact, patients have been misdiagnosed, and their physicians advised of this misdiagnosis, which subsequently is recorded on death certificates. Dr. Whitehouse, in his report to this court, indicates that in his mortality study of 79 deaths from *"asbestosis"* (his definition), 46% had an ILO chest x-ray reading of 0/1 or less, i.e., they had no evidence of asbestosis. The result of the use of the term *"asbestosis"* as a

wastebasket to describe even minimal pleural plaquing makes comparisons with other populations, in which the term is accurately and correctly defined, confusing and utterly meaningless.

23. A radiographic survey of more than 7,000 current and former residents of Libby and the surrounding area was performed between 2000 and 2001 (Peipins, 2003). The study was supported by the Agency for Toxic Substances and Disease Registry. The participants included former W.R. Grace workers, household contacts of W.R. Grace workers, and persons with environmental exposures but no occupational exposures. Less than 1% of the screened population had interstitial abnormalities consistent with (but not diagnostic of) asbestosis. The rate of interstitial abnormalities in males increased from 0.04% in adults up to 44 years of age; to 0.5% for participants between 44-65 years of age; to 3.2% greater than 65 years of age. With rare exceptions, the ILO readings did not exceed 1/1 (personal communication, Spence, M.). Males had a higher rate of interstitial abnormalities (1.2%) than females (0.4%). Current or former smokers were twice as likely to have interstitial abnormalities as those who had never smoked.

24. The diagnosis of asbestosis, as stated by the American Thoracic Society 2003 position statement, requires the satisfaction of three main criteria. First, there must be evidence of structural pathology consistent with asbestos-related disease as documented by imaging or histology (appropriate chest x-ray changes and/or pathological changes). Second, there must be an appropriate history of exposure and an appropriate latency period consistent with the development of asbestosis. Third, and importantly, there must be exclusion of alternative plausible causes for the

findings (Jones, 1991). Diseases or conditions that may mimic asbestosis include congestive heart failure, pulmonary vasculitis due to rheumatoid arthritis or systemic lupus, usual interstitial pneumonitis or idiopathic pulmonary fibrosis, crowding of the vascular markings in the lung bases secondary to poor inspiratory effort, and accentuation of basilar lung markings secondary to obesity. As pointed out by Gaensler (1991), "it is crucially important not to miss a potentially treatable disease before the onset of irreversible fibrosis, and not to assume that all interstitial lung diseases in asbestos-exposed workers are asbestosis."

25. There is a general agreement from epidemiologic studies that the development of asbestosis requires heavy exposure to asbestos and that there is a threshold fiber dose below which asbestosis is not seen (Churg, textbook). A number of studies suggest that this dose is in the range of 25-100 fibers/cc-years. Accordingly, asbestosis is usually seen only in individuals who have had many years of high-level exposure. The latency period for the appearance of asbestosis is inversely proportional to exposure level (Becklake, 1983). Fiber burden studies indicate that the lungs of workers with asbestosis consistently contain the highest fiber levels seen in any of the asbestos-related diseases (Roggli, 1986). An expansion of that earlier study, published in his 2004 textbook, (Roggli, textbook) indicate a median asbestos content in lung tissue of patients with asbestosis of 152,000 uncoated asbestos fibers per gram of wet lung, as compared to a median content of 14,800 fibers per gram in patients with pleural plaques only.

26. The prevalence of asbestosis, usually defined by the presence of small irregular opacities on the chest x-

ray and by a profusion score 1/0 using the ILO classification, is highly variable according to the type of fiber, type of industry, cumulative exposure history, and age. Epidemiological studies involving 33 different cohorts between 1971 and 1994 show prevalence ranges of 2-16% in asbestos miners (chrysotile contaminated by tremolite or crocidolite), 7.5-40% in workers manufacturing insulation products, and 0.8-60% in end-product uses (Hendrick D, textbook, 2002). The highest prevalence was found among a cohort of 2790 insulation workers with chrysotile and amosite exposure (Lilis, 1991).

27. The radiographic abnormalities of asbestosis may progress beyond the time when exposure ceases, but the rate of progression is usually slow. Overall, progression is observed in less than 50% of cases after exposure cessation, depending on the exposure conditions, the initial radiographic profusion score, and the interval between radiographs (Jones R, 1989; Rubino G, 1979; Gaensler E, 1990; Jakobsson K., 1995; Becklake M, 1979).

E. Pleural plaques

28. Pleural plaques are scars or markings on the outer lining of the lung. Viewed at surgery, they look like drops of candle wax. Pleural plaques are discrete circumscribed areas of thickening of the parietal pleura (outer lining of the lung) of the chest wall and diaphragm. Plaque formation appears to be one of the common ways in which the pleura reacts to injury, since pleural plaques have been seen as a long-term sequel to infection, particularly tuberculosis; trauma (including chest surgery); and hemopneumothorax (Churg A,

textbook). They are also the most common respiratory effect of asbestos inhalation, occurring at asbestos exposure levels far below those required for the development of asbestosis (Roggli V, textbook). Asbestos-related pleural plaques are characteristically bilateral, although not symmetric, and most typically occur on the lower portions of the parietal pleura and the diaphragm. Over time, pleural plaques may become extensively calcified.

29. With rare exceptions, focal circumscribed pleural plaques cause no symptoms. They are generally noted as an incidental finding on chest film.

30. Pleural plaques as an isolated radiological finding do not by themselves produce clinically significant reductions in pulmonary function (Jones R, 1988). Published studies purporting to demonstrate an adverse effect of plaques on pulmonary function (Whitehouse A, 2004) have not adequately controlled for confounding effects of other factors, such as cigarette smoking. A study in which the surface of localized pleural plaques was quantitated by computed tomography scanning (Van Cleemput, 2001) found no correlation between cumulative asbestos exposures and the size or total surface area of pleural plaques. Importantly, there was no significant correlation between either the presence or the extent of pleural plaques and lung function. These findings are in agreement with the opinion that moderate pleural plaques, in the absence of other asbestos-related disorders, have little or no effect on lung function tests (Hillerdahl G., 1985; Mossman B, 1989; Jones R, 1988;)

31. As stated in the chapter on benign asbestos-related pleural disease in Roggli's textbook, Pathology of Asbestos-Related Diseases, "The lack of symptoms or signs in the majority of patients with pleural plaques alone

leads one to ask whether pleural plaques should be considered a disease."

F. Asbestos Pleural Effusion and Diffuse Pleural Thickening

32. The term diffuse pleural thickening (DPT), which is a less common manifestation of asbestos exposure than pleural plaques, relates to thickening that does not have well-circumscribed margins and that may involve 25% or more of the surface of the lung (Miles S, 2008). DPT is the result of extensive fibrosis of the visceral pleura with adhesions to the parietal pleura. It may be unilateral, but is more often bilateral. In one large study of asbestos-exposed insulators (Bourbeau, 1990), DPT was relatively rare (5.5%) as compared to pleural plaques (52.5%). Although DPT of a limited extent may be an asymptomatic incidental finding on chest film, more extensive disease may encase the lung and impede expansion, resulting in shortness of breath with exertion.
33. Prospective studies of asbestos workers have shown DPT to occur in between 5 to 13.5% of workers (McLoud TC, 1985; Bourbeau J, 1990).
34. Most experts believe that diffuse pleural thickening is the result of an episode (or recurrent episodes) of benign asbestos pleural effusion. By a mechanism that has not yet been elucidated, asbestos exposure may result in acute inflammation of the pleural space with or without a radiologically detectable pleural effusion. The effusions are generally small, unilateral, transient, and asymptomatic. However, in one study recurrent effusions developed within ten years in 28%, sometimes on the same side but more frequently on the opposite side (Epler G, 1982). The resultant organization of the inflammatory

effusion is thought to result in diffuse fibrotic changes of the visceral pleura with eventual development of radiologically visible DPT. All the patients observed by Epler developed obliteration of the costophrenic angle as the effusion organized. The inclusion of costophrenic angle blunting for the diagnosis of DPT by the 2000 ILO, which Dr. Whitehouse finds so upsetting, is the result of the acknowledgement of the relationship between benign asbestos pleural effusions and the development of DPT.

35. Significant diffuse pleural thickening results in a restrictive defect on pulmonary function testing. FVC and FEV1 are reduced proportionately, as is total lung capacity and DLCO (carbon monoxide diffusion). It is important to note that the reduction in DLCO is primarily the result of reduction in lung volume rather than a derangement of alveolar tissue. Consequently, DLCO corrected for alveolar volume will be normal (Rudd, 1996).
36. Longitudinal studies of patients with diffuse pleural thickening have indicated little if any progression in loss of pulmonary function. It is felt that the occurrence of DPT is accompanied by an initial restrictive loss of lung function followed by relatively stable pulmonary function in the majority of cases (Yates, 1996).
37. The diagnosis of asbestos-related diffuse pleural thickening is a diagnosis of exclusion. The differential diagnosis includes tuberculosis, previous chest trauma (especially hemithorax, previous surgery such as coronary artery bypass grafting, recurrent pleurisy secondary to pneumonia, rheumatoid arthritis and other connective tissue diseases, fibrosing pleuritis, post-radiation therapy, and the use of certain drugs such as methysergide. Without high-resolution CT scanning, it if often difficult to distinguish extrapleural fat in an obese population from pleural thickening.

G. Pulmonary function tests

38. Pulmonary function tests (PFT's) are a frequently used tool in the diagnosis and differentiation of various lung disorders.
39. Although many different measurements can be made with sophisticated testing equipment and computer programs, there are six that have the most clinical relevance: FVC, FEV1, FEV1/FVC ratio, TLC, RV, and DLCO. These values must be looked at as a set for an accurate and meaningful interpretation.
40. Forced Vital Capacity (FVC) is a measurement of the volume of air that can be expelled after taking as deep a breath as possible and blowing it out as fast and as long as possible. The amount of air that is expelled in the first second of expiration is the Forced Expiratory Volume in one second (FEV1). The ratio of FEV1 to FVC (FEV1/FVC) is normally about 0.8.
41. Total lung capacity (TLC) is a static measurement of the total amount of air in the lungs. It is most accurately measured with a plethysmography (body box). The amount of air remaining in the lungs after complete exhalation is the residual volume (RV).
42. The ability of oxygen to be transferred from the lung to the bloodstream is estimated by the carbon monoxide diffusion test (DLCO). The exchange takes place across what is normally a one-cell thick membrane between the microscopic oxygen-containing unit of the lung, the alveolus, and the pulmonary capillary. Oxygenated blood is then carried to the left heart where it is pumped to the vital organs.

43. A patient with advanced asbestosis (scarring of the interstitial lung membranes) is most likely to show the following pattern of pulmonary function abnormalities: Because his lungs are diffusely scarred, they are stiff. Consequently, it is not possible to take as deep a breath as a person with normal lungs. As a result, both the FVC and the FEV1 will be reduced. However, they will be reduced proportionately, and the FEV1/FVC ratio will remain normal. Both the total lung capacity and the residual volume will be reduced. Because there is scarring and thickening of the membrane where oxygen is delivered to the blood, the DLCO will be reduced.
44. A patient with severe diffuse pleural thickening (without underlying asbestosis) would be expected to have normal lungs encased in a fibrous “girdle” that prevents full expansion of the lungs. As a consequence, the FVC and FEV1 would be expected to be proportionately reduced with preservation of a normal FEV1/FVC ratio. Total lung capacity and residual volume would be reduced with preservation of a normal RV/TLC ratio. DLCO would be expected to be reduced, but only as a function of encased alveoli. Consequently, correction of the DLCO for alveolar volume (DLCO/Va) should result in a normal value.
45. Patients with chronic obstructive pulmonary disease (COPD) generally have some combination of two distinct disease processes: chronic bronchitis, which involves chronic inflammation and swelling of the airways in addition to hypersecretion of mucus, resulting in cough and phlegm; and emphysema, which involves destruction of lung tissue, frequently leaving holes in the lung that can be seen on CT scan of the chest. In many patients, a combination of these processes is evident. In others, bronchitis or emphysema may be the dominant pattern of disease.
46. Both chronic bronchitis and emphysema result in an obstructive pattern on pulmonary function testing, but by

slightly different mechanisms. In bronchitis, the bronchial tubes are narrowed, swollen, and often mucus-filled. Blowing air out through these tubes can be compared to exhaling through a soda straw with the nose plugged. It is a more difficult and slower process than blowing air through normal-diameter bronchi.

47. In emphysema, there is destruction of lung tissue to the point that “Swiss cheese-like holes” (bullae) can often be seen on CT scan. This destruction leads to loss of elastic recoil of the lungs. A normal lung functions like a new toy balloon. If it is blown up and released, air is vigorously expelled because of elastic recoil. An emphysematous lung, however, functions like the balloon that has been blown up, tied off, and allowed to sit in the corner for six weeks. When the tie is removed, a small amount of air will escape, but primarily external pressure will be required to expel air from the flabby balloon. With the flabby lungs of emphysema, significant amounts of air may be trapped in the lungs and external muscles of the neck and chest wall must be used to force air out significantly slowing the process of exhalation.

48. Consequently, a patient with COPD may show the following patterns of pulmonary function abnormalities: With less advanced COPD, the patient may have a normal FVC, but the FEV1 and the FEV1/FVC ratio will be reduced. As COPD increases in severity, the FVC may also become reduced. Total lung capacity may be normal. However, as COPD increases in severity, air trapping may increase with consequent elevation of the TLC and the residual volume. Importantly, the ratio of residual volume to total lung capacity is often increased. In patients with emphysema, large portions of the membrane between alveoli and pulmonary blood vessels have been destroyed. *Consequently, decrease in DLCO is prominent feature of emphysema.*

49. Patients may have a mix of obstructive pulmonary disease from smoking and a restrictive defect from severe kyphosis of the chest wall that may occur with aging or from pulmonary fibrosis resulting from gastroesophageal reflux and scarring of the lung tissues. Under those circumstances, all of the pulmonary function data must be critically examined by an experienced PFT reader for accurate interpretation.
50. When one piece of data from pulmonary function testing is extracted and discussed separately from the tests as a whole, as when Dr. Whitehouse states, “We find that the DLCO defect is the leading indicator of severity in the Libby cohort,” the conclusions are at best meaningless and at worst, deceptive. Without being able to look at the test as a whole, we are unable to tell if the patient with decreased oxygen diffusing capacity has restrictive disease or emphysema. Likewise a “decrease in the FVC” is meaningless without knowing the FEV1 and the FEV1/FVC ratio, since FVC can be decreased in both restrictive and obstructive disease.

G. Asbestos airways disease, chronic obstructive pulmonary disease, and tobacco smoking

51. It has been my personal experience that patients diagnosed as having “asbestosis,” instead have chronic obstructive pulmonary disease secondary to tobacco smoking.
52. Dr. Whitehouse states in his report to this court that only 15% of smokers develop clinically significant chronic obstructive pulmonary disease. In fact, we have long known that 86.9% of men with a history of smoking up to one pack of cigarettes per day had evidence of emphysema at autopsy, as compared to 99.7% of those who had smoked more than one pack per day (Auerbach, 1972). Of those

smoking less than one pack per day, 25.1% had moderate emphysema and 11.7% had advanced emphysema. Of those smoking more than one pack per day, 32.7% had moderate emphysema and 19.2% had advanced emphysema. In addition, rates of chronic bronchitis among smokers have been estimated at 45-70% (Speizer, 1979).

53. The development of chronic obstructive pulmonary disease is related to individual susceptibility (most likely genetic differences) as well as the age smoking was initiated and the cumulative smoking history measured in pack-years (number of packs of cigarettes per day X number of years of smoking). It is not uncommon in my Montana practice to see patients in their forties who have already accumulated smoking histories in excess of 75 pack-years from having started smoking two to three packs of cigarettes per day by age twelve.

54. Inhalation of asbestos, in addition to a number of other minerals, can produce fibrosis of the walls of bronchioles (small airways). Asbestos airway disease has been described by pulmonary pathologists (Wright, 1992) as fibrosis and distortion of the airway wall, sometimes accompanied by the presence of asbestos bodies. In his report, Dr. Whitehouse strongly implies that asbestos airway disease is the same entity as chronic obstructive pulmonary disease. It is not.

55. Asbestos airway disease can be demonstrated to cause changes in parameters that are only measured in research laboratories, such as increased isoflow volume and increased upstream resistance (Begin, 1987). These parameters, although of interest to researchers, are of no clinical relevance. Begin studied 34 lifetime nonsmoker asbestos miners and millers (exposed to tremolite) and found that although asbestos airway disease could be shown to cause airflow limitation at low lung volumes, it did not reduce the FVC or FEV1. He demonstrated that in

smoking asbestos workers with asbestos airways disease or asbestosis, the major component of airflow limitation is a smoking effect.

56. These findings are confirmed in a study of Chinese asbestos workers (Wang, 2006) in which multivariate regression analysis showed that decreased ratio of FEV1/FVC (indicative of obstructive airways disease) was best predicted by smoking amount, whereas neither asbestos exposure nor asbestosis resulted in a decrease in the FEV1/FVC ratio, strongly suggesting that smoking, rather than asbestos, was the major factor responsible for airway limitation in those asbestos workers.

57. As stated in Hendricks' textbook, Occupational Disorders of the Lung, (Hendricks, 2002) "asbestos is not currently thought to contribute to chronic obstructive pulmonary disease (COPD) and there is no evidence for a role of asbestos in producing emphysema. When COPD is detected with asbestosis, it is likely to be coincidental and a consequence of cigarette smoking."

58. The "obstructive pattern associated with Libby amphibole asbestos disease" as described by Dr. Whitehouse, is in fact chronic obstructive pulmonary disease secondary to tobacco smoking, as illustrated by the following examples.

59. Mr. M is a 66-year-old man seen in October 2008 at the request of the Libby Medical Program for a second opinion regarding the etiology of his pulmonary disease. Mr. M gave a history of having smoked two to three packs of cigarettes per day from age 22 until he was hospitalized with acute respiratory failure in 2002, giving him an estimated cumulative smoking history in excess of 90 pack-years. He has been disabled and oxygen-dependent since 2002. He requires continuous oxygen at 3 liters/minute at rest and at 4 liters/minute at night. He lived in Troy but primarily traveled all over the country in his work as a core driller. He never worked in the vermiculite mine

or mill in Libby. He did run a heating and refrigeration business in Libby for about ten years. Prior to his illness, Mr. M weighed 160-170 pounds. With the inactivity imposed by his respiratory disease, he has progressively gained to 287 pounds and has developed Type II diabetes mellitus. He has a history of obstructive sleep apnea (OSAS), but has been unable to tolerate the CPAP device used to treat OSAS. Consequently he is at high risk for oxygen desaturation at night despite his supplemental oxygen. An echocardiogram has shown severe chronic cor pulmonale (right heart failure) with marked dilatation of the right atrium and right ventricle and suspected pulmonary hypertension. Left ventricular function was normal. CT scan of the chest showed a few scattered focal pleural plaques. There was no evidence of diffuse pleural thickening. Severe, bilateral bullous emphysema was noted with the majority of the lung tissue replaced by large holes. There was no evidence of bibasilar interstitial disease consistent with asbestosis. Pulmonary function testing performed at the Center for Asbestos Related Disease in June 2007 showed an FVC of 79% predicted, an FEV1 of 38% predicted and an FEV1/FVC ratio of 0.38. Total lung capacity was 150% predicted with a residual volume of 309% predicted and an RV/TLC ratio of 202% predicted. Lung diffusion was severely reduced, consistent with his emphysematous disease, at 27% predicted. *Mr. M has severe, end-stage chronic obstructive pulmonary disease and chronic cor pulmonale secondary to a long history of tobacco abuse. His respiratory problems have been compounded by the development of morbid obesity and obstructive sleep apnea. Although he has CT scan evidence of scattered focal pleural plaques, asbestos exposure has in no way caused or contributed to his severe lung disease.* Nevertheless,

both he and his wife appear to be convinced that all his medical problems are “from Libby asbestos.”

60. Ms. J is a 62-year-old woman who presented in April 2008 for a second opinion on her lung disease. She was a resident of Libby for thirty-seven years. She worked in the lumber mill for about five years, “pulling and pushing lumber.” She also did some waitress work, but was primarily a housewife. Ms. J smoked two packs of cigarettes per day from age 14 until age 58, for an estimated cumulative smoking history of 88 pack-years. For two months prior to her visit, she had experienced progressively increasing shortness of breath with exertion. Two days prior to her visit, she developed a severe cough productive of green mucus and was treated with antibiotics and supplemental oxygen. On examination, she was a kyphotic elderly lady with hyperinflated lungs, severely diminished breath sounds, and scattered end-expiratory wheezes. Her fingernails were cyanotic. She had pitting edema to the knees bilaterally. Four sets of chest films from 2004 to 2008 were reviewed, as were two CT scans of the chest (2001 and 2008). Both plain films and CT scans revealed minimal focal calcified pleural plaques at the lung bases bilaterally. There was no blunting of the costophrenic angles. There were chronic shaggy interstitial markings in the left lung base consistent with post-inflammatory scarring. There was no evidence of asbestosis. Pulmonary function studies showed an FVC of 42% predicted. The FEV1 was 29% predicted with an FEV1/FVC ratio of 0.56. Total lung capacity was 109% predicted with a residual volume of 216% predicted. Lung diffusion was diminished at 41% predicted. These findings are consistent with severe obstructive airways disease with hyperinflated lungs, air trapping, and decreased DLCO secondary to emphysematous changes. *In summary, Ms J who is being followed in the CARD clinic for “asbestosis” has*

severe chronic obstructive pulmonary disease with evidence of right heart failure, acute bronchitis, and hypoxemia. She has some focal pleural plaques as indicators of asbestos exposure, but no evidence of asbestosis. Her asbestos exposure has not caused or contributed to her lung disease. Fortunately for Ms J, we were able to recommend some measures to hopefully improve her functional status.

61. Ms. N is a 74-year-old WF who had requested a second opinion regarding her lung disease. She was seen in July 2008. She had presented to the CARD clinic four months prior to her visit with complaints of gradually increasing shortness of breath with exertion. She was determined to be hypoxemic and had been started on low-flow continuous oxygen in addition to a Serevent diskus and Spiriva Handihaler, with significant improvement in her symptoms. Ms. N has lived in Libby since birth. She had never worked in or around the mills or mines. She had smoked one pack of cigarettes per day for 35 years, with smoking cessation thirteen years ago. On examination, she had a kyphotic chest wall with evidence of hyperinflation of the lungs. Breath sounds were distant without wheezes, rales, or rhonchi. A recent CT scan of the chest revealed scattered calcified and non-calcified focal pleural plaques but no evidence of asbestosis. Pulmonary function testing showed an FVC of 83% predicted, an FEV1 of 60% predicted, and an FEV1/FVC ratio of 0.56. Total lung capacity was 103% predicted with an elevated residual volume of 133% predicted, indicative of air trapping. Lung diffusion was diminished at 39% predicted, consistent with loss of alveolar-pulmonary capillary interface secondary to emphysematous changes. *Ms. N is an elderly lady who is followed at the CARD clinic for "asbestosis." CT scans reveal pleural plaques as evidence of asbestos exposure, but no evidence of asbestosis. She has*

moderately severe chronic obstructive pulmonary disease related to her history of cigarette smoking, and may have a mild superimposed restrictive defect secondary to her kyphotic chest wall.

H. Lung cancer

62. Lung cancer has not been discussed by Dr. Whitehouse in his report. However, because I am diagnosing and treating lung cancer in Libby residents and CARD clinic patients, I believe a discussion is relevant to these proceedings.
63. Cigarette smoking is by far the most common risk factor for the development of lung cancer. It is estimated to cause at least 95% of lung cancer cases in the United States.
64. The idea that any exposure to asbestos, no matter how small, results in an increased risk of lung cancer is not supported by epidemiological studies (Weiss, 1999). In fact, prospective studies in both asbestos cement workers (Hughes, 1991) and amosite miners (Sluis-Cremer, 1989) have demonstrated an increased risk of lung cancer only in the presence of *asbestosis,* (not pleural plaques). The presence of pleural plaques does not mean it is more likely than not that a person will develop cancer. It should be noted that lung cancer in non-smoking workers with asbestosis is rare. As of 1995, Morgan and Seaton (Textbook) had found only thirty case reports of lifelong nonsmokers with asbestosis and lung cancer.
65. Since the incidence of accurately defined asbestosis is low in the current Libby population, one would not expect a significant increase in the incidence of lung cancer over that of the smoking population.
66. To date, I have seen five patients from Libby and/or the CARD clinic with lung cancer. The majority of them had

pleural plaques, but none had a history of occupational exposure to vermiculite and or radiological evidence of asbestosis. All had cigarette smoking histories that placed them at high risk for lung cancer without invoking any other possible risk factors.

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